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EDITORIAL

BOOKS TO-DAY AND TO-MORROW

LORD TEMPLEWOOD, when speaking in the House of Lords lately, did well to focus attention on by far the greatest destruction the Nazis have wrought on mankind. Great as is the material destruction, incalculable as is the injury to human health and development, both are as nothing to the mental and moral degradation of mankind. No one, before the Hitler gang, ever conceived so great a wickedness as the training of the young from childhood days in every form of moral perversion : no one has so thoroughly made vice virtue and virtue vice : no one has so deliberately bred a generation efficiently equipped morally to perform every kind of bestiality towards any nation or individual. They have spared their own kith and kin no less than their neighbours.

Lord Templewood was timely in his warning to mankind, and his words will reach a much wider audience than the House of Lords. He speaks at a moment when our nation, which stands for truth, kindliness and forthright honesty, finds one of the most potent weapons it possesses to combat the machinations of the Nazis, lying limp and weak in its hands.

Books are the bulwark of freedom, and without them all progress and all human endeavour are frustrated. But books are notoriously scarce, books of all classes. Our school children are going without their class-books, our undergraduates without their text-books. The case is particularly serious in regard to medical and veterinary students. Turn to the scientist, the research worker and the industrialist, and the same lamentable shortage is apparent. England has a glorious account to give of herself in the past years and millions of books are wanted in every field and walk of life, but there does not appear to be any prospect of their being published.

The Publishers' Association has just issued a memorandum entitled "Medical Books—the inadequacy of their supply in relation to present and future requirements." This memorandum covers veterinary books of all kinds, and the eye is at once arrested by two words in the title, "inadequacy" and "future." They bring one up with a jolt. Most of us with complacency console ourselves by saying: "Well! you know, there is a war on," and we permit that to account for the deficiencies of the present. This infers that the future will be infinitely better and that all our troubles will melt away when the war is over. But what is the position? Turn to the medical publishers and hear what is their considered finding. In an accompanying letter to their memorandum they say (the italics are ours): "*There can be no prospect of any improvement in this serious situation unless and until the Government makes more paper and more labour in the printing and binding trades available for books.*" The memorandum concludes with these words:—

> "To sum up, the special problems connected with the production of medical books demand full and early consideration. Medicine cannot stand still without injury to the whole community; equally, it cannot advance without books. It is consequently most urgent that adequate measures should be taken to improve the supply of medical books, for even if the existing difficulties could be remedied to-day, many months would pass before any appreciable improvement would be apparent."

If the medical publishers are correct, and their opinion should carry great weight, they are trying to explain to the veterinary profession—and to each and every member of it—that not only are veterinary books, and those of allied science, in grievous and injurious short supply, but that there is no prospect of early remedy, nor are there any plans in being to effect an improvement at some future date.

Here surely is a matter of grave concern to our profession, and one to which the Royal College of Veterinary Surgeons and the National Veterinary Medical Association should give immediate attention. Representations should be made to the appropriate Government authorities urging them to set about caring for the supply of veterinary books. Yes, "caring" is the apt word, for if a man does not care, he does not bother. To numbers of people, books may be no more than a relaxation, but to the veterinary surgeon they are an essential and indispensable working tool, the restriction of which inflicts widespread injury on the community, and a long-term injury at that. We, therefore, trust that the leaders of our profession, and especially those who teach, will impress on the Government the prime necessity of ensuring an adequate supply of books, for books are the very breath of life. It is an embittered and demoralised world which lies before us, and there is no means of eradicating hatred and dispelling misunderstanding, but by the full and free dissemination of knowledge. Our appeal, though primarily concerned with our own needs, goes much further, for in the interests of the nation and of the whole world, the freest possible distribution of the largest number of books is a first essential of the peace to come, and now is the time when the necessary plans must be made.

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General Articles

CHEMICAL WARFARE AND FARM ANIMALS*

By MAJOR W. P. BLOUNT, R.A.V.C.

Introduction

CHEMICAL warfare (C.W.) means gas and all its allied implications. This paper also includes a note on the hazard of phosphorus poisoning in warfare.

One of the most important points to grasp at the outset is that there is often a narrow margin between the amount of gas which will injure an animal seriously and that which does little harm. Therefore the actual amount of polluted air breathed is of vital importance. This in turn is related to the state of the animal at the time, i.e., resting, walking, running, etc. For example, a galloping horse inspires about 10 times the amount of air compared with one at rest, and would therefore be receiving 10 times the "dose" of gas, and this could obviously tip the balance between life and death adversely.

In Great Britain it is rare for the weather to be such that there is no wind, and therefore in moving out of a gas cloud it is essential to move upwind—i.e., to travel against the wind.

If these two points are borne in mind they will prove of real value on the farm should it be subjected to gas attacks.

Type of Attack

Farmers may expect gas attacks by plane, and such aircraft may either spray liquid gases or use bombs which explode just before reaching the ground (air-burst).

The effect of an air-burst bomb, filled with a liquid, would be similar to that which occurs with low spray from an airplane.

Bombs may carry any type of gas, and may be any size from 1 to 1,000 kilos.

Type of Gases

The two main methods for classifying gases are:—

(a) *Tactical*

So far as is known Germany has not produced any C.W. agent specially for attack on animals, so that gases are classed tactically by their use on man.

(1) *Lethal*—designed primarily to kill, quickly. *Example* prussic acid.

(2) *Casualty producing* but capable of causing death if sufficient is absorbed, e.g., mustard.

(3) *Harassing*, designed to render a soldier temporarily incapable of his war effort, e.g., tear gas.

The tactical use of these gases against animals would be similar, except that the harassing agents do not affect animals, or, to be effective, must be used

* Being the substance of a lecture delivered recently at King's College, Newcastle-on-Tyne, to animal stewards, farmers and veterinary surgeons.

in such high concentrations that this would not ordinarily be possible under field conditions.

(b) *Pharmacological*

The action of a chemical agent on an animal frequently differs from its effect on man. For example, as may well be known to many, the vesicants (e.g., mustard and lewisite) do not cause blisters on animals.

Furthermore, there are differences within the species. It is well known that canaries are more susceptible to carbon-monoxide poisoning than other common animals, not because they are small but because they are sensitive to its lethal effects. Hence the use of canaries in coal mines for detecting choke-damp or after-damp. Similarly there is a distinct difference between man and dog concerning prussic acid. The experiment of Sir Joseph Barcroft during the last war, in which he showed that he was unaffected by prussic acid, whereas a dog died, may be recalled in this connection.

Casualties Among Farm Stock

A classification of the casualties likely to occur amongst farm stock is not easy, because of the differing functions of the animals concerned. For example, a horse (rider) need not be classed as a casualty because he has been burnt about the belly with mustard, providing he can still carry his rider from place to place, whereas a dairy cow similarly affected might be useless for milk production owing to udder damage. Similarly, a young gilt might not be inconvenienced by a spot of lewisite on the belly, whereas in this situation a boar could suffer readily from retention of the urine.

The main causes for casualty in animals are :—

(1) *Blindness*—temporary or permanent; e.g., vesicants.

(2) *Burning of the Skin* to involve a vulnerable area, such as the heel of the horse, udder of cow, teats of sow, feet of sheep and dog; e.g., vesicants.

(3) *Respiratory Distress*—constant coughing, broncho-pneumonia, pulmonary œdema, etc.; e.g., chlorine, phosgene.

(4) *Affections of the Digestive System*—
   (a) Ulceration of the mouth or stomach; e.g., vesicants.
   (b) Colic; e.g., phosphorus poisoning.
   (c) Diarrhœa; e.g., mustard poisoning.

(5) *Systemic Effects*—
   (a) Redwater; e.g., following arsine poisoning.
   (b) Anæmia; e.g., following mustard poisoning.
   (c) Asphyxia; e.g., following CO and prussic acid poisoning.
   (d) Shock; e.g., following lewisite poisoning.

One or two special points arise concerning animal casualties relative to their natural protection, behaviour and anatomy. Firstly, the coat of the horse, cow and dog, the wool of the sheep and the feathers of the hen and pigeon give quite good protection against mustard droplets, whereas the pig—comparatively hairless—suffers much more severely. In this respect, since lewisite is a much

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more fluid "gas" than mustard, it runs between the hairs and wool easier and does more damage to the skin. The action of lewisite generally is more violent, so that such lesions are often painful and bloody (hæmorrhagic), best seen in the pig. Feathers give quite good protection even against lewisite.

The fact that dogs lick their feet when irritable renders them specially susceptible to the systemic (general) effects of mustard-lewisite, and an efficient muzzle may be a necessity for the prevention of such accidents.

Animals with long tails (e.g., the horse and cow) are a danger to themselves as well as to their owners if contaminated by vesicants, because with one swish they may spread the liquids concerned to other and more vulnerable areas, including the attendant's eyes, face or hands.

Finally, there is the example of the hen's wattles. These are richly supplied with blood vessels, and if a drop of lewisite falls on them it soon becomes volatile. This vapour rises quickly to affect the eyes, in the form of a series of spasms readily noted as "blinking."

The horny layers of an animal's body, beak, hoof, claw, etc., offer complete resistance to penetration by mustard, etc., but the coronary band—the horn-producing layer at the top of the foot—is not so protected and is readily damaged by lewisite.

Gas Effects on Farm Animals

For practical purposes it is best to consider only two types of gases—the vesicants and the choking gases, whilst the special hazard of phosphorus poisoning is also worth reviewing.

Vesicants

Mustard and lewisite are typical vesicants. They do not cause blisters on animals, probably because the many hairs of the natural coat anchor the epidermis to the dermis and so successfully prevent their separation to form a blister. But under the influence of vesicants these same hairs become erected (especially in the warmer months of the year), and this provides quite a useful warning sign that liquid gas has fallen on the coat. The "burn" produced by mustard quickly becomes prominent to the touch due to a fluid infiltration (œdema), but this subsides after 24 hours, and for 3-4 days one can merely feel a lump beneath the hairs; this somewhat resembles a "warble." Each drop of vesicant produces its own "burn," of course, so that there may be dozens along the animal's back. Next the "scab" so formed cracks at the edges and eventually is thrown off, though it may remain as a "*sitfast*" for several weeks. The underlying scar is soft at first and may not bear weight (as with saddlery or harness) for 2-3 months after the onset of the original injury.

Lewisite is more mobile and penetrates the hairs more easily than mustard, its odour is characteristic (geranium) and its effects more severe, though the healing time is somewhat shorter than with mustard. It is an immediate irritant even on the skin and causes a burn characterised by the destruction of blood vessels locally so that, if cut into, the affected part shows much hæmorrhage as well as œdema.

A lewisite burn on the horse separates quicker than with mustard, but follows the same type of healing process.

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On sheep the mustard burn is red, whereas that with lewisite is mauve-purple, a contrast even more marked in the pig and especially well seen after 2-3 days. (The pig's ear occasionally does show a shallow bleb or blister at the site of vesication and is thus the exception to the rule.)

In the mouth the affected parts are covered by a false membrane which may quickly ulcerate to resemble superficially a foot-and-mouth lesion. Salivation is prominent and healing slow, the breath stinks and in ruminants there is grinding of the teeth. The appetite may fail completely, especially if there is added damage to the stomach and intestines, when the excreta is loose and sometimes bloody.

In the *eyes* the vesicants do irreparable harm, and the blindness which follows application of the tiniest drop of mustard or lewisite may remain permanently.

Lameness is marked when the vesicant has fallen at the back of the knee, front of the hock, over the tendons or between the toes. In dogs the toes may be exposed to the bone and even slough away.

Apart from their direct action, the vesicants give off an irritant vapour. This is serious for the skin of man, especially if hot and sweaty. Fortunately, the action on the skin of farm animals is much less serious, but its inhalation in prolonged or large amounts causes nasal and eye discharges, bronchitis and pneumonia.

First-aid Treatment for Vesicant Gases

The eyes are so sensitive to vesicant gases that they need first-aid attention before the remainder of the body. It is preferable, therefore, to have a team of four people working, one to secure the animal, one washing out the eyes, one decontaminating the remainder of the body and a fourth preparing more bleach or getting the next animal to hand.

First-aid Treatment for the Eyes

For *mustard,* liquid B.B.C. (tear gas) and allied substances, a thorough washing of the eyes with water is a necessity. This must be done as early as possible after contamination, but it is doubtful whether the addition of salt or bicarbonate of soda (one teaspoonful to a pint) is of real value.

The eyes of animals cannot be washed satisfactorily—i.e., so that the fluid penetrates fully between the lids—unless a simple glass or bakelite nozzle is attached to the rubber tubing used. The cork of the bottle holding the fluid should have a nick down one side, so that air can enter to displace the water as it runs out. The washing should be continued for several minutes.

For lewisite an effective treatment is available and will no doubt be issued by the authorities should such an emergency arise.

General Decontamination

(1) Remove all visible contamination by means of cotton waste, cotton-wool, cloth, sacking, bags, leaves, grass, etc. (Harness and saddles are taken off as early as possible.)

(2) Prepare bleach cream 1 : 3 in water and apply freely. *Never use dry bleach owing to the risk of fire.*

(3) Remove the bleach thoroughly in 10 minutes by washing the parts freely in water. Dry the coat. Rug up if necessary.

(4) In the absence of bleach, anti-gas ointment, hydrogen peroxide or potassium permanganate, use soap and water or water alone, freely. For decontamination of the feet and legs in an emergency take the animal to a running stream. To walk through long wet grass is also quite useful. Note, however, that this spreads the contamination.

(5) The interval between contamination and the application of the bleach cream or other decontaminant should be as short as possible. Decontaminants are of little value to the animal after one hour, but are necessary in any case to neutralise gases which would otherwise spread to involve other animals or attendants, i.e., unabsorbed gas on the hairs, tail, and hoof.

(6) Report the matter to your warden.

Treatment proper is essentially a matter for the veterinary practitioner.

Choking Gases

The best example of a choking gas is *phosgene.* It differs from chlorine in that it does not cause coughing when first inhaled, and has a latent period during which few recognisable symptoms appear. It damages the lower parts of the respiratory system, i.e., not the nose, voice box, windpipe or large bronchial tubes, but the lungs, and the animal "drowns" due to the severe outpouring of fluid into the air sacs. As a symptom of this in the horse, masses of straw-coloured froth pour from the nose an hour or so before death.

First-aid Treatment for Choking Gases

(1) Remove the animal to a shed where there is fresh (uncontaminated) air as early as possible, but do not do so except at a walk.

(2) Occupy its mind by providing food, e.g., steamed hay.

(3) Rug it up and protect it from inclement weather.

(4) Report the matter to your warden.

Treatment.—As with the vesicants, skilled veterinary attention is necessary for the after-treatment of animals suffering from the effects of choking gases.

Prevention

The prevention of gas casualties is much easier and more effective than treatment, and is broadly divisible into that which concerns the inhaled gases and that for the liquids.

Respirators and Allied Protective Measures

There are horse and dog respirators in use in the Army to-day.

For *horses* an improvised respirator can be made quickly by filling a nose-bag with wet (damp) hay, drawing this over the nose and taking the strap over the poll and fastening it so that it is secured firmly. If the respirator so made covers only the upper jaw (nose) it will be just as effective as if it is made to enclose both the nose and mouth. In the latter case the nosebag should only be filled two-thirds with hay and then covered with a cloth to prevent the horse

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eating it. After this is applied to cover the nose and mouth more wet hay is stuffed around the nose and jaw to complete the filling of the nosebag. For *cattle* the nosebag should cover the nose and mouth as just described.

In the absence of any such protection, reliance should be placed upon making a stable or byre gas-proof. This does not necessarily mean that it is to be hermatically sealed, but that as much air as possible is to be prevented from entering by natural means.

This automatically means sealing up all windows, doors, cracks, ventilators, air bricks, drain pipes, etc.

Special attention should be paid to those near the floor level or any entering the building near the head of the animal (e.g., Tobin's tube). Ridge ventilators and those high up by the eaves should be attended to last of all.

In a sealed room it might be thought that animals would soon suffocate, but two experiments we have carried out show how false is this assumption :—

(a) A dog weighing 30 lb. occupied a completely air-tight chamber, approximately 3 ft. cubed, for three hours with little or no discomfort.

(b) A horse occupied a specially constructed air-tight stable 12 ft. x 12 ft. x 12 ft. (1,730 c. ft.) without ventilation for over 24 hours and suffered no discomfort.

In the case of the horse the body temperature and pulse remained normal, whereas the respiratory rate increased slightly from 16 to 20 respirations per minute. The relative humidity of the stable rose to 90 per cent., and there was water of condensation dripping from the roof corrugations. The atmosphere was close and clammy, but not unbearable. The horse had eaten two feeds of corn, a quantity of hay, and consumed 1½ buckets of water in the 24 hours.

Theoretically a horse should be able to withstand such conditions for 36-48 hours, so that such a simple method of protecting farm stock is particularly valuable. As a fact, non-persistent gases of the phosgene variety under all ordinary conditions would be dispersed in a matter of minutes and ½-1 hour should see even the highest local concentrations dissipated by natural means. Farmers may therefore arrange their animal buildings so that in an emergency every air inlet can be completely blocked, knowing full well that, if necessary, their stock will remain healthy overnight.

Protection Against Liquid (Sprayed) Gases

This concerns eyeshields, clothing, overhead cover and ointments.

Eyeshields are excellent for protecting the eyes against sprayed gases and in the Army are in use for horses and mules.

To avoid sprayed gases it is only necessary for animals to remain indoors or to be afforded *overhead cover* in some form or other. A covered yard, outbuilding or barn is all that is necessary to give adequate protection. Hurdles may be used on the prevailing wind side to add further protection against sprayed blister gases. If there is any choice of buildings in the housing of stock, those not situated in valleys or *culs-de-sac* are best; outbuildings on high or rising ground with protection on the weather side are ideal, especially if a water point or stream is near at hand. For all practical purposes protection from rain is the equivalent of protection against gases (sprayed liquids).

When it is necessary to move animals short distances, e.g., from one building to another, or from place to place, and contaminated ground has to be crossed, a watery bleach mixture should be sprayed in the form of a footpath and covered with a thick layer of straw or hay. Animals may then cross the area with comparative safety. Straw alone will suffice if no bleach is to hand. Again note that *dry* bleach must not be used, because the heat produced when in contact with mustard and allied gases is so great a fire may easily be started, especially near a stackyard.

It should never be necessary for animals to leave a building whilst gas spraying is in progress, because the latter only lasts at the most a matter of 30 minutes. If it were absolutely necessary, *clothing* of some sort should be worn, such as rugs, sacking, blankets, etc.

If contaminated ground must be crossed by farm stock, protect the feet and legs by stable bandages, sacking (sandbags) or other cloth tied in position, but for preference first anoint the hollow of the heel or cleft between the toes with *anti-gas ointment.* Remove this as early as possible and decontaminate as previously advised.

The attendant must see that he himself is protected, preferably by the use of A.G. ointments, before handling contaminated stock.

Bleach

The quantity required may be calculated at 2 lb. per large adult animal, i.e., 1 cwt. drum per 50 head of stock.

Anti-gas Ointment

One-quarter the amount of bleach should be purchased, i.e., 28 lb. per 50 animals.

Detectors

A variety of detectors for the various gases will be available should C.W. break out, but they may not be discussed now.

Protection of Foodstuffs

The protection of foodstuffs on the farm against liquid gases is not difficult, because any under cover are automatically protected, and the non-persistent gases, such as phosgene, can be removed simply by airing the fodder in the sun or wind.

Drinking water contaminated by vesicants is dangerous to use and should never be drawn from stagnant supplies—ponds, etc. That from a running stream or deep well is usually safe, as well as piped supplies. All water of a doubtful character should be boiled 30 minutes. Water containing arsenical gases is highly poisonous and cannot readily be purified.

Milk known to be contaminated by liquid gas is unsafe, but if it is only affected by vapour it should be boiled 15 minutes before use.

Crops

After contamination by a non-persistent gas these should be allowed to remain a few days before use, but in general fodder which can be aired freely is

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usually safe, providing there is no trace of foreign odour and no danger of arsenical poisoning.

Crops freshly contaminated by mustard and lewisite are dangerous to handle or use; the latter gas kills many crops mainly because of its high arsenic content. Mustard retards the growth of plants but they recover later, except possibly in the case of sugar beet, which is killed.

Phosphorus Bombs and Farm Animals

Incendiary and other bombs containing phosphorus offer two separate and important hazards for farm stock:—

(1) Burns.
(2) Poisoning.

Phosphorus Burns

The long hairy coat of horses and cattle offers some protection to burning phosphorus particles, but the resulting burn is identical in character, though reduced in size, when compared with that which occurs in clipped or short-coated animals. Where the heat is greatest all the hairs are burnt away, whilst others in the vicinity are singed. The central portion of the burn is hard and tough, like dry leather. There is no discharge, but a zone of swelling (œdema) surrounds it. After several days or weeks the scab comes away, though surgical means may be necessary to complete the process. The underlying area is fibrous and frequently painless. The healing process is very slow indeed. The phosphorus is not absorbed into the system to any extent and there is no danger of phosphorus poisoning.

First-aid Treatment

Copper and bicarbonate solutions are not necessary for farm stock—a cold wet compress applied as early as possible is excellent to soothe the parts and remove the charred hairs and debris. In our experience, elemental phosphorus particles burn themselves out completely and quickly, and no ordinary specific (e.g., copper containing) remedies have been of value in assisting recovery from such burns.

After-treatment should be left to the discretion of the veterinary practitioner.

Prevention

Afford protection as for high explosives.

Phosphorus Poisoning

Shells or bombs containing white phosphorus—e.g., smoke producing weapons—after their explosion and ignition do not render their full complement of phosphorus inert, and as a result contamination of pastures or crops by certain toxic phosphorus compounds occurs. This offers a serious risk of poisoning to all farm stock, including poultry.

Fortunately the weather reduces this risk relatively quickly, and, in general, 2-3 weeks after such contamination has occurred the phosphorus is neutralised by rain and wind.

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Symptoms of Phosphorus Poisoning

The horse may be taken as a typical example. One or two days after eating the phosphorus-contaminated pasture signs of colic appear. These are of renal (kidney) origin and become intensified, the animal dying within 24 hours. *Post-mortem* findings include gastritis and inflammatory lesions throughout the urinary system. The large intestines are normal. In ruminants, on the other hand, the kidneys are normal, but the "coiled" colon is severely damaged with blood-stained, constipated contents. The walls of the "stomachs" are inflamed or sloughing. The well-known advanced fatty degeneration of the liver is common to all species of animals.

Treatment

This is essentially a matter for the qualified veterinary surgeon.

Prevention

Do not allow animals to graze suspected land for 21 days after any incident involving phosphorus. Remove empty shell or bomb cases to a place of safety.

THE URETER AND ARTERIAL BLOOD SUPPLY TO THE KIDNEY OF THE CAMEL
(Camelus bactrianus)

By PROFESSOR T. GRAHAME,
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A TEN-YEAR-OLD female camel (*C. bactrianus*) was destroyed in the gardens of the Zoological Society, Edinburgh, 1944.

The kidneys were removed at the post-mortem and the right kidney was taken for anatomical examination. The left kidney was injected with celloidin to demonstrate the arteries and ureter.

The writer has not been able to find any work published on this subject. Any references to the anatomy of the camel's kidney are inadequate and have no bearing on this type of work.

The most helpful treatise is the "Anatomy of the Urogenital System of Mammals" by Morison. This work is useful in that a definite nomenclature has been applied and an endeavour has been made to maintain this terminology throughout. The references given in THE VETERINARY JOURNAL, Vol 100, No. 5, May, 1944, on "The Kidney of the Indian Elephant," will serve as a guide to the technique and general descriptive methods. The same technique which was used to inject the kidney of the elephant was applied to the kidney of the camel with equal success. Perhaps the finer vessels are better injected, which makes the reading of the cast much easier.

Ureter

The hilum affords an exit to the ureter and an entrance to the arteries. Immediately the ureter enters the hilum there is a sharp constriction of the tube, which gives the appearance of the presence of a sphincter muscle. The

bore of the tube at this point is reduced to almost half its diameter. Beyond this constriction the tube is gradually dilated to form a cone-shaped tunnel which suddenly expands into what is appreciated as the pelvis of the kidney. This dilated cephalic extremity is designed to receive the fused renal papillæ of the renal pyramids. From the coalescence of papillæ a renal crest is evolved. It appears that the disposition of the renal pyramids in the kidney of the camel is similar to the arrangement of the pyramids in the kidneys of sheep and dog. The formation of a renal crest is clearly demonstrated in the sections of the right kidney. The crest occupies a sagittal position and extends the length of the kidney, but does not appear to have great width, in spite of the convergence of the lateral renal pyramids to it. A detailed description of the renal pelvis is necessary to understand its curious appearance. The free margin of the pelvis seems to adopt the buttress design as noted in the pelvis of the sheep and dog. The free edge is interrupted by fissures which isolate well-defined areas, the buttresses of the pelvis. Although each buttress has in general a similar shape and extent, each one has its own individual character. There are eight on each side of the pelvis, with one at each extremity. Each buttress is enfolded with a fluted and crenated margin. In some the edges enfold, almost meeting, to constitute a canal-like structure. The fissures on either side of the buttress are irregular and of varying width, although the depth appears to be consistent. The folded-over walls of a buttress form the lower wall of a fissure and the upper wall of the next fissure, and it is therefore apparent that two buttresses are necessary to complete the boundaries of a single fissure. Where the sides of the buttresses meet at the lower margin of the fissure a broad flange is created which overlaps the funnel of the pelvis. The free edge of the flange is crenated and it terminates in a point oriented towards the hilum. The buttresses are mostly single and are almost tubular in appearance, but occasionally one splits into two tubular-like structures with a comparatively short V-shaped fissure between them. The free extremities of the buttresses extend laterally towards the cortex and often abut those from the opposite surface and occasionally overlap. All the buttresses are not confined to the surface from which they arise, but may extend across the median plane to the other side. Two buttresses meeting end on give the impression of continuity. Independence is clearly revealed on closer examination. The fissures between the buttresses are in communication with one another over the ends of buttresses and there is a direct communication between the fissures on either side. This has been indicated by the opposition of the buttresses. A probe may be passed directly through from one surface to the other without interference. The end buttresses are single, large and massive, with a fissure on either side. This formation gives these buttresses an independent character. It will be appreciated from the above that the buttresses and fissures have a definite and regular arrangement.

The inference must therefore be that the pyramidal tissue of the kidney is more or less dependent upon the primary structures of the metanephric dilation to accommodate the metanephric tubules. Therefore it must be concluded that the renal pyramids are also disposed in a regular and definite plan. Several of the fissures are almost divided into two equal-sized recesses. This is effected

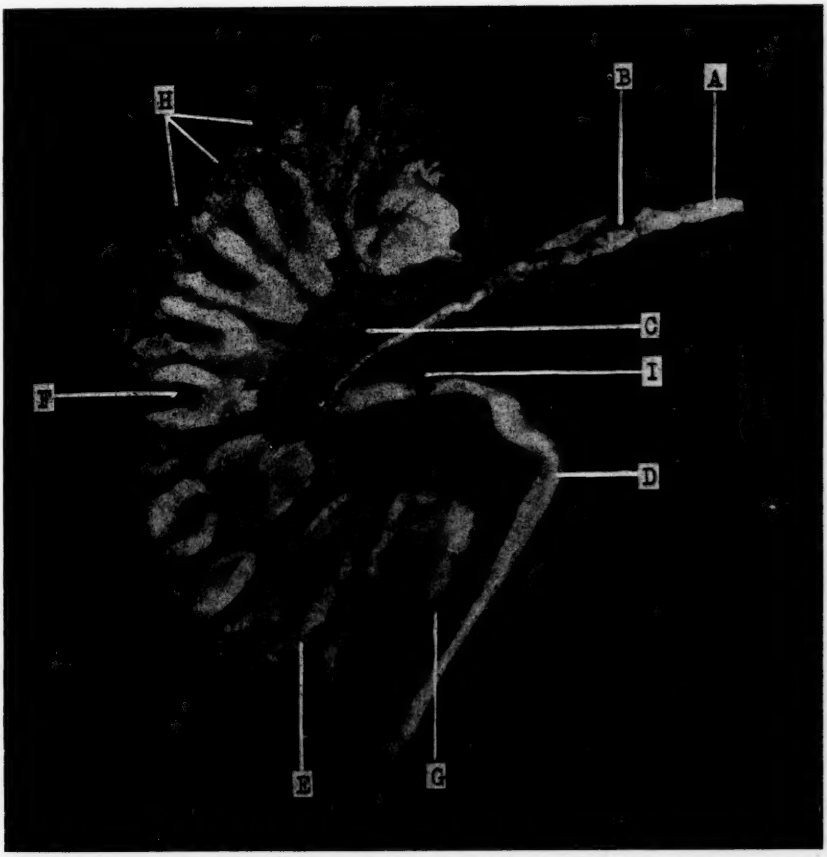

PLATE I

FIG. 1.—A photograph of a celluloid cast of the arteries and ureter of the kidney of a camel. (½ original size).

A—Renal artery. B—Ventral and Dorsal branches. C—Interlobar arteries. D—Ureter. E—Buttress of pelvis. F—Fissure. G—Arcuate artery. H—Interlobular arteries. I—Artery to ureter and pelvis.

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PLATE II

FIG. 2.—An enlargement of part of Plate I to illustrate more clearly certain vessels.
A—Arcuate artery. B—Interlobular vessels. C—Nutrient vessels to the medulla.

by the lateral boundaries of the fissure being evaginated by an artery given off on either side of the fissure. If the arteries so given off happen to be immediately opposite one another the evagination of the edges of the fissure are almost in contact, giving the double character to the fissure.

It is assumed that the fissures provide the route for the renal pyramids to gain access to the central recess of the pelvis, where they meet to constitute the renal crest. The buttresses give support to the larger vessels and the crenations of their margins support to the smaller vessels.

Arteries

A short distance from the aorta the renal artery divides into a dorsal and a ventral branch. Between these two main branches the ureter lies, and in this formation they enter the hilum of the kidney.

Dorsal Artery.—This vessel enters the hilum on the dorsal aspect of the ureter. Upon reaching the pelvis it curves backwards to the terminal buttress. The dorsal artery is superficial, with the convex border towards the cortex, from which arise the interlobar branches.

Ventral Artery.—The ventral artery lies below the ureter. It has a similar course and distribution to the dorsal vessel. The first branch to be given off from the dorsal artery is a small one, which appears to supply twigs to the ureter and the apical part of the pelvis. Following this branch a series of interlobar vessels arise from the convex side of the vessel.

The Interlobar Arteries.—Before the branching of interlobar vessels a small vessel breaks away from the dorsal artery to supply the ureter and part of the pelvis. The dorsal vessel gives origin to eight interlobar arteries. The first interlobar vessel divides into two branches before reaching the pelvis. A branch goes to each gutter of the first and second buttress. The first buttress divides into two parts and therefore the cephalic branch also divides, giving a branch to each division of the buttress. The second buttress is undivided, as is the artery which is the caudal branch, or second interlobar vessel. The subsequent buttresses and vessels are undivided. The second branch to arise is the third interlobar vessel, which enters the third groove directly opposite its site of origin. The fourth, fifth, sixth and seventh interlobar vessels originate in a similar manner to the third and have a similar course and distribution. The eighth interlobar vessel is the terminal part of the dorsal artery, and it follows the gutter formed by the buttress at the cephalic end. The groove formed wholly by the caudal buttress is also supplied by an interlobar branch from the ventral artery.

The interlobar arteries within the groove give origin to many arcuate vessels and each interlobar vessel terminates in the formation of one or more arcuate vessels. It is interesting to note that no arcuate branches are given off, except in the grooves.

The Arcuate Arteries.—The arcuate vessels are distinguished, as they are in the kidneys of other mammals, by their size, shape, course and divisions. As the arcuate vessels emerge from the gutters they usually groove the edge of the buttresses, giving them a crenated appearance.

Many of the arcuate vessels run in a parallel course, more especially those derived from the same interlobar vessels. Most are parallel to the surface of the kidney. Some arcuate vessels from adjacent interlobar vessels anastomose. It would not be surprising to find a continuous anastomoses between the surface arcuate arteries of each interlobar vessel.

The number of arcuate arteries which arise from each interlobar vessel appears to vary in accordance with the area to be supplied. The arcuate vessels, which are derived from the interlobar artery within the gutter of the buttress, vary considerably in their length, course and branching.

(a) A short type which do not issue from the gutter but form a tuft of interlobular capillaries which spread out over the edges of the laminæ of the buttresses.

(b) A larger type which come on to the surface where they bend, to form the characteristic arch. The convexity of the arch is more abrupt and shorter than the arch of the arcuate vessels given off nearer the end of the buttress. These vessels are noticed mostly midway along the fissures, over which they bend, breaking up into a brush of fine interlobular capillaries. These interlobular vessels behave differently from the usual plan, they run parallel and not at right angles to the surface. Each fissure is thus circumscribed by a rich capillary network. Many of the terminal branches supply the medulla.

(c) Near the end of the buttress several arcuate vessels are seen to emerge, each with a short curved course parallel and close to the surface, and each vessel giving a complete mass of interlobular capillaries which intermingle, forming a complex network in the cortical tissue. There does not appear to be any anastomoses of this group of vessels. There are no branches to be seen coming from the concave side of the vessel either within or without the gutter of the buttress. These terminal arcuate vessels contribute no ramifications to the medullary area, all their supply is cortical.

(d) The first branch from the interlobar artery, just within the commencement of the groove, bends medially towards the medulla into which it gives a profuse aborescence of small vessels.

Interlobular Arteries.—The interlobular arteries are numerous, short, and create the delightful network of the surface.

These arteries pass out from the arcuate vessels at varying angles. Near the termination of the arcuate vessels they appear to be at right angles to the parent vessels, but further back the angles are more acute. The interlobular artery may remain single, or undergo several sub-divisions. The latter method is more common. There is a gradual attenuation from the arcuate vessels to the interlobular vessels, and these, although they may sub-divide, do not become much reduced in calibre. All the interlobular vessels are more or less of similar length and diameter.

The interlobular arteries terminate in diverse ways.

(a) To run a short straight course towards the subcortical tissue and constitute the afferent of a glomerulus.

(b) To give off several short afferents, each of which forms a glomerulus. The terminal twig either remains as a single filament or divides into two small filaments. The latter is more common. Each filament becomes the afferent of a glomerulus.

(c) The interlobular vessels frequently divide, forming tufts of small nutrient capillaries of the cortex.

(d) Some of the stronger interlobular vessels pass right through the subcortical tissue and perforate the capsule, constituting the perforating capsular arteries, to the perirenal tissue.

Summary

(1) The buttresses and fissures of the pelvis are characteristic of unilobar kidneys, i.e., dog, cat, sheep, etc.

(2) The dorsal and ventral arteries, branches of the renal, which give rise to a series of interlobar vessels, which constitute a simple means of adequately supplying the renal pyramids.

(3) Special arcuate vessels appear to give only nutrient capillaries to the medulla and do not form glomeruli.

(4) The absence of straight capillaries from the concave side of the arcuate arteries.

(5) The presence of perforating capsular arteries.

(6) A small nutrient artery to the infundibulum of the pelvis and ureter.

(7) The scheme of renal arterial distribution conforms to the general plan, as observed in mammals.

I am greatly indebted to Mr. P. G. D. Morris and Mr. William Neill, of the Histology Department, Royal (Dick) Veterinary College, Edinburgh, for their assistance and care in the production of the model and the very excellent photographs.

MARIE'S DISEASE ASSOCIATED WITH TUBERCULOSIS IN A HORSE

By E. COTCHIN,

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Introduction

MARIE'S disease (syn.: hypertrophic pulmonary osteoarthropathy, acropachia, hypertrophic pulmonary osteoperiostitis, Cadiot's syndrome, etc.) presents in animals a clinical picture of a slow, progressive, and usually symmetrical increase in size of the limbs due to the production of fibrous tissue and subperiosteal bone. It is generally associated with some other chronic condition, particularly of the lungs. The condition in animals most often associated with Marie's disease is tuberculosis, but the bone changes themselves are apparently not due to the direct action of the tubercle bacillus. Any part of the skeleton may be involved, but usually the limb bones are chiefly or only affected. The condition has been recorded in animals most often in the dog, but has also been

seen in the cat, ox, deer, lion, horse and fowl. Occasional cases in the dog have been recorded in this country (Smythe, 1929; Wooldridge and Holmes, 1937; Innes, 1940; Craig and Davies, 1940; Cotchin, 1944).

Review of Literature on the Disease in the Horse

Marie's disease is apparently not common in the horse, and only a few records occur in the literature. A case described by Chambers (1891) is possibly the first recorded. He saw a tuberculous colt in which the vertebral bones were covered with "a deposit of bony matter," due, he surmised, to a "scrofulous ostitis." His article was not illustrated, however, so that the first undoubted case would seem to be that of McCall (1902), who gives photographs showing the typical lesions on the bones; he called the case one of "multiple exostoses." An aged bay carriage gelding lost condition, showed albuminuria, and developed a progressive œdema of the hind limbs, sheath, and abdomen. Within six months the horse became stiff in the hind limbs, and the bones began to show swellings. At post-mortem examination the only general change found was anæmia. There were exostoses on the limb bones, the chalky crumbling outgrowths being confined to the periosteum; the underlying bone and bone marrow appeared normal. McCall commented on the uniformity of the outgrowths and the absence of involvement of the articulations.

Liénaux (1909) saw a case in a tuberculous horse, describing the lesions as a diffuse osteoperiostitis. It is possible that the condition demonstrated at a practitioners' meeting by Ison (1916) was also a case of Marie's disease; he found changes in various bones in a tuberculous mare. Jarmai (1926) recorded a case in a horse. The lesions were discovered when the animal was slaughtered, and were associated with a general senile atrophy of the organs. In his article is a photograph of the bony growths on the pelvis, femur, tibia, humerus and metacarpus. Histologically the periosteum was thickened, with a rich inner layer of osetoblasts; between the periosteum and the compacta was a broad zone of the new bony tissue, the peripheral part of which was formed of closely-packed trabeculæ, those in the centre being looser in arrangement. These new trabeculæ were arranged roughly parallel to each other, and vertical to the compacta, with a sharp line of demarcation between the compacta and the new bone. The compacta was rather porous, but the bone marrow appeared normal. No inflammatory changes were seen in the old or new bone, and inoculation of ground-up material from the lesions did not produce tuberculosis in guinea-pigs.

Wirth and Pommer (1931) saw a case in an eight-year-old light gelding which ate well but lost condition. The hind limbs were affected first, showing œdematous swellings up to the mid-tibial region, a change of consistency to bony-hard taking place in the course of six or seven weeks; later, the fore limbs showed similar changes. X-ray pictures showed radially arranged new subperiosteal bony trabeculæ, not involving the joints. The lesions were most obvious on the shafts of the radius, tibia and cannon bones, and three small lesions were found on the lower border of the left horizontal mandibular ramus. At the *post-mortem*, small calcified nodules were found in the lungs, and interstitial fibrosis, but guinea-pig inoculation tests were negative for the presence of

tubercle bacilli in the lung. Nietsche (1933) saw a case of Marie's disease in a horse, in which the hind limb changes developed in about three months, but the fore limbs were not affected until about two and a half years later. A tuberculin test was positive, but tuberculosis could not be demonstrated *post-mortem*. Kulczyki (1933) described the disease in a horse bearing an infected fibroma. Maly and Czernok (1933) recorded a case of what they took to be Marie's disease in a nine-year-old horse, but the restriction of the bony changes to the phalanges and distal ends of the metacarpals, and absence of lesions elsewhere, led von Guoth (1938) to suggest that this may not have been a true case of the disease. Von Guoth himself described a few cases of the disease (1937, 1938, 1939, 1940). In two of these, young animals, otherwise in good condition, were affected. He found a tendency, so far not reported by other authors, for the condition to regress spontaneously. Jarmai and Móczy (1941) saw a tuberculous horse affected with Marie's disease.

CASE REPORT

A 3 to 4 years old gelding had suckled until it was nearly a year old, had never been off grassland, and was unbroken. Although it had never been given cow's milk, the gelding was running on land on which bullocks had been grazed for the past twenty years, and it is possible that this was the source of the tuberculous infection found later. The animal was observed to hold its head in a forward position, and all four limbs showed swellings of bony consistency extending from the knee or hock to the fetlock. The appetite was good, and the animal was able to rise and lie down normally. It was suspected during life that the gelding was affected with Marie's disease, and in view of the frequent association of that condition with tuberculosis, tuberculin tests using avian and bovine tuberculin were performed. A positive reaction was obtained to the bovine tuberculin, and it was decided to destroy the animal. Blood smears taken at the time of death indicated the presence of anæmia and neutrophilia: Oppermann and Doenecke (1933) mention the association of leucocytosis and erythropenia with tuberculosis in horses. The blood clotted normally.

POST-MORTEM FINDINGS

A. Lesions Other Than of the Bones

Macroscopic Findings.

There was subcutaneous œdema, especially over the abdomen and around the upper joints of the limbs. A little brownish turbid fluid was present in the body cavities. The mesenteric, para-aortic and mesocolic lymph nodes were enlarged, firm and nodular: the mesenteric nodes formed a mass measuring 18 in. by 12 in. by 12 in., composed of about twenty nodules of varying size. These nodes showed a fibrous capsule, a brownish vascular subcapsular zone, and a pale yellow rather firm centre, in which no obvious foci of necrosis, caseation or calcification were found. Acid-alcohol fast organisms were present in small numbers in smears from the mesenteric nodes. The swollen nodes around the abdominal aorta had apparently pressed upon it to cause an increased peripheral resistance to the blood flow, as the left ventricle of the heart was

greatly hypertrophied. There was a slight fibrinous deposit on various serous membranes. The liver substance appeared normal, but the hepatic lymph node was greatly enlarged (5 in. by 4 in. by 3 in.) and similar in appearance to the mesenteric nodes. The spleen was not enlarged, but showed in its substance, and in some places bulging the capsule, about fifty small, rather firm, pale yellowish-white irregular nodules up to ½ in. diameter. The hilar nodes were enlarged up to 2 in. diameter. The bronchial lymph nodes and a mediastinal lymph node were also affected, but there were no obvious lesions in the lungs. A lesion of strongyle ulceration was found in the cæcum. There was an increase of fibrous tissue in the limbs, and some œdema around the flexor tendons.

Microscopic Findings.

Histologically, the bronchial lymph node showed typical equine tuberculous granulation tissue. In the lungs were very small scattered foci of cell-infiltration of the interstitial tissue, possibly tuberculous in nature. The liver revealed a diffuse amyloid change in sections, and very small tuberculous foci were present. The spleen, besides the tuberculous foci, showed many small areas of amyloid change. In the kidneys there was some hyaline material in the capsular spaces and tubules, with cloudy swelling and some fatty changes in the tubular epithelium: no amyloid changes were seen.

B. Lesions of Bones

Skeletal lesions were present on the mandible, the vertebræ and the limb bones.

Mandible.

The right mandible at the angle of the jaw showed a smooth swelling up to about twice its normal thickness; the borders of the swelling faded gradually on to the normal bone. In sections, the mandible was seen to be ensheathed by a regular subperiosteal layer of radially arranged almost parallel bony trabeculæ (Fig. 1). This layer was thickest at the lower border of the mandible (Fig. 2) and showed here a dense structure of well-formed Haversian systems. Passing up the mandible, the layer of new bone grew evenly thinner, the trabeculæ becoming narrower and more widely separated by a scanty fibrovascular tissue. The lesion merged smoothly into normal periosteum (Fig. 3). The line of demarcation between the newly formed and the original bone was sharp. The mandible itself showed a slight degree of osteoporosis, particularly around the marrow cavity.

Vertebræ.

The vertebræ showed nodular exostoses on the posterior part of the left surface of the superior spinous process of the axis, just anterior to the posterior articular process, on the ventral aspect of the body of the axis just behind the anterior articular process, and on the right ventrolateral surface of the body of the sixth dorsal vertebra. The body of the latter bone was also swollen and projected downwards as a nodule 1½ in. in diameter into the thoracic cavity, but not eroding the pleura. In section of the sixth dorsal vertebral body, two types of change were seen—a tuberculous osteitis and an extensive subperiosteal formation of new bony trabeculæ. The latter were similar in appearance to

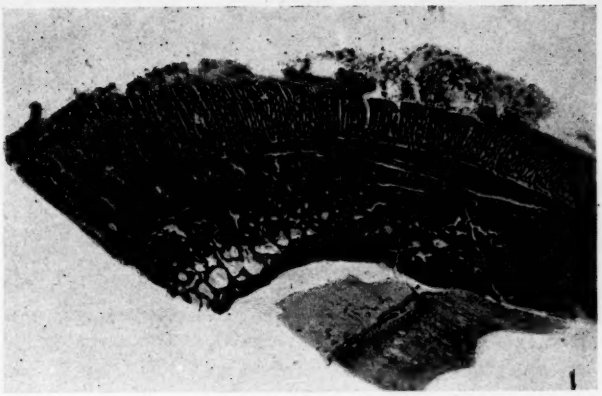

FIG. 1.—Part of lesion of angle of mandible, showing the newly formed periosteal trabeculæ of bone (above) and their roughly parallel arrangement. X 3.

FIG. 2.—Part of the same lesion, showing gradual diminution in density and thickness of the periosteal new bone towards the upper margin of the lesion (left). Note in this and the previous section the osteoporosis in the mandibular ramus, and the sharp line of demarcation between the new and original bone. X 3.

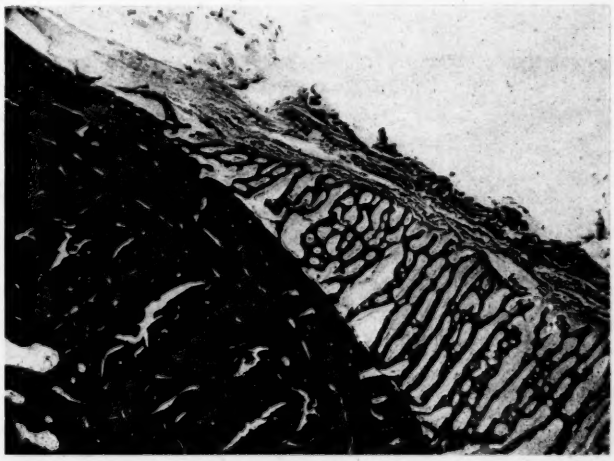

FIG. 3.—Part of mandibular lesion, showing merging of abnormal and normal periosteum at the edge of the lesion, and the arrangement of the newly formed trabeculæ. X 18.

XUM

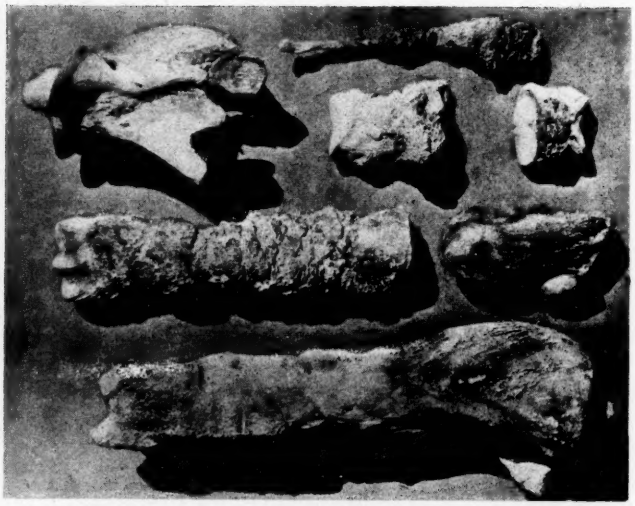

FIG. 4.—Macroscopic appearance of some of the bones showing the periosteal bone deposits.

FIG. 5.—Section of metacarpal bone, showing the original compact bone (right) and the newly formed periosteal bone (left). The latter has a looser structure in the outer half. X 3.

(*All sections stained by Haematoxylin and van Gieson*)

those seen on the mandible, but small foci of tuberculous granulation tissue were present in the intertrabecular spaces, both next to the body and towards the raised periosteum, and around these foci the trabeculæ had either disappeared or had not been formed. The large number of new trabeculæ, their regular character, and the small number of tuberculous foci, gave the impression that the tuberculous process in the subperiosteal region was superimposed upon the periosteal change, and did not precede it. Such a secondary infection of the previously formed exostoses may account for those cases on record (e.g., Sendrail and others, 1920) where tubercle bacilli have been found in the limb bone lesions in cases of Marie's disease.

Limb Bones.

Most of the limb bones showed bony exostoses, and some are shown in the photograph (Fig. 4). The lesions were most evident on the metacarpal, metatarsal and phalangeal bones, and on the tibia. In no case were articular surfaces involved. In sections of a metacarpal lesion (Fig. 5), the periosteum was thickened, and overlay newly-formed trabeculæ of bone, which in some cases had a roughly parallel arrangement. The trabeculæ near the compacta were quite thick and closely packed, the superficial ones thinner and more widely separated by a loose fibrovascular tissue. In some parts, only the deeper layer of denser bone was present. No tuberculous foci were seen in sections of the metacarpal bone.

C. Bacteriological Examination

A portion of the bronchial lymph node was emulsified in sterile saline and treated with oxalic acid (5 per cent. final concentration) for 40 minutes; tubes of plain egg and glycerin egg media were then inoculated with the treated material and incubated at 37° C. Colonies of acid-alcohol fast organisms were visible on the plain egg, but not on glycerin egg, three weeks later. The colonies were umbonate, with a rough peripheral growth, and moderately dry in appearance, although they were emulsified fairly readily.

Guinea-pigs and rabbits were inoculated with an untreated bronchial lymph node suspension in saline, with the following results:—

Guinea-pig 1.—Received 1 c.c. suspension subcutaneously in the right thigh, 28/1/44. When killed, 14/4/44, the precrural lymph node was enlarged (1 cm. diameter) and contained yellow caseo-pus. The inguinal lymph node (0.5 cm. diameter) showed a similar change. No other obvious lesions were seen. Smears of the nodes mentioned showed acid-alcohol fast organisms to be present, and a dysgonic culture resembling that recovered from the horse was obtained on egg medium after five weeks' incubation. No organisms were detected by smear or culture in the spleen.

Guinea-pig 2.—Similar dose, 28/1/44. This animal was killed in excellent condition on 29/6/44. A chain of caseous nodules was found extending from the site of inoculation to the inguinal lymph node, which was 2.5 cm. in diameter and filled with yellow caseo-pus. No other lesions were seen. Smears showed a few tubercle bacilli to be present in the inguinal node, but none was seen in smears from the spleen.

XUM

Rabbit 1.—A young rabbit received 1 c.c. of lymph node suspension subcutaneously over the left shoulder on 28/1/44. It died after prolonged wasting on 15/5/44. There were several small caseous foci at the site of inoculation, and half a dozen tubercles in each lung. Smears and cultures showed the presence of a dysgonic strain of tubercle bacillus.

Rabbit 2.—An adult rabbit received 5 c.c. of lymph node suspension subcutaneously over the left shoulder on 28/1/44. It was destroyed in excellent condition on 29/6/44. There were about twenty pinhead-size tubercles at the site of inoculation, in which a moderate number of tubercle bacilli were present, but there were no other obvious lesions.

These results suggest that the type of tubercle bacillus present in the lesions in the horse was a dysgonic strain of very much reduced virulence for guinea-pigs and rabbits, of the kind described by Griffith (1928).

Discussion

The cause of Marie's disease in animals remains to be determined. Although so frequently associated with tuberculosis, it is also seen in connection with other conditions, such as neoplasia and anæmia, or even as an apparently primary disease, and it is permissible to postulate some underlying metabolic derangement as the cause of the bony changes in these varying circumstances.

Although a parallel arrangement of the trabeculæ of newly formed subperiosteal bone has been described in this and other articles on Marie's disease, it is not known whether it is present in all cases. It seems that this particular type of periosteal reaction is not pathognomonic, since Theiler (1934) figures a similar arrangement of trabeculæ in an exostosis on a rib of a horse with osteodystrophia (his Fig. 24), and a further example is seen in a photograph by Kodiceck and Murray (1943) of sections of hyperostotic lesions of the tibia and fibula of guinea-pigs subjected to a prolonged partial deficiency of vitamin C. In the latter instance, the authors suggested that the lesions might be caused partly by weakening of the collagen fibres attaching the periosteum to the bone and partly by subperiosteal hæmorrhage, both of which would allow the periosteum to separate from the bone surface. The situation, appearance and delimitation of the lesions of the bones in Marie's disease suggest the possibility that here also the periosteum is raised from the bone, as, for example, by œdematous fluid, so that the fibres and vessels lying on the bone surface are drawn out to a position somewhat perpendicular to the surface, new bone then being formed alongside these fibres and vessels.

Summary

(1) The literature on Marie's disease in the horse is reviewed.
(2) A case of Marie's disease in a tuberculous horse is described.

Acknowledgments

The case was first examined by Major J. Sheffield, M.R.C.V.S., who kindly provided the clinical details. The horse was given to the Royal Veterinary College by the owner, in view of the interest of the case. The pathological examination was made at the request of Prof. Formston, F.R.C.V.S.

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Abstracts of Current Literature

SURVIVAL OF INFECTIVE LARVÆ OF HÆMONCHUS CONTORTUS*

LARGE numbers of larvæ of the common stomach worm of ruminants (*Hæmonchus contortus*) were exposed outdoors on grass plots for four seasons at the Animal Disease Research Station, Beltsville, Maryland. The plots were examined at intervals of two weeks. In the summer, the larvæ recovered dropped to about 1 per cent. of the inoculum after 14 to 28 days; in the fall, after 40 to 56 days; in the winter, after 27 to 41 days; and in the spring, after 56 to 70 days. No larvæ were recovered after exposures of 42, 83, 98 and 316 days during the summer, winter, spring and fall, respectively. These figures represent minimum estimates of the survival of the larvæ outdoors. A lamb that grazed for nine days on two plots, containing infective larvæ which had been exposed for 266 days, did not become parasitised. Of all the larvæ recovered in the four experiments, 43 per cent. were found on the grass, 24.2 per cent. on surface soil, 32 per cent. in the first inch of soil, and 0.8 per cent. in the second inch of soil. The horizontal migrations were comparatively slight.

* *American Journal of Veterinary Research,* January, 1944.

News

VACCINATION AGAINST CONTAGIOUS ABORTION

FOR the past two and a half years two live vaccines, No. 1 vaccine made from the American Strain 19 and No. 2 vaccine prepared from the English Strain 45/20, have been approved for use in T.T. herds and herds participating in the Scheme for the Control of Diseases of Dairy Cattle which were known to be infected with contagious abortion.

The available evidence suggested that both vaccines would be effective when used in adult animals, No. 1 vaccine in a single dose and No. 2 vaccine in two separate doses. No. 1 vaccine had also been widely and successfully used in the United States of America for the vaccination of calves and was accordingly adopted for that purpose in this country. One slight disadvantage of No. 1 vaccine is that inoculated animals develop agglutinins soon after injection and will therefore react to the blood agglutination test. Such animals usually become normal again, however, in the case of vaccinated calves before the time of first calving. No. 2 vaccine, on the other hand, was not thought to stimulate the production of agglutinins and was therefore considered to be the more suitable vaccine for adult animals in herds from which animals are sold on a negative agglutination test certificate.

The reason for restricting the use of the two vaccines to infected herds in the first instance was that full information as to their stability in the tissues of injected animals was not available. In the interval further experiments have been carried out, the results of which justify the extension of the use of No. 1 vaccine to any female bovine animal of any age, whether or not the herd is infected with contagious abortion. Accordingly, female animals in any T.T. herd in England and Wales or in any herd participating in the Control Scheme may now be vaccinated with No. 1 vaccine.

Experiments with No. 2 vaccine, on the other hand, suggest that it may undergo changes in inoculated animals which in some cases may cause them to react to the agglutination test, that it is less effective than No. 1 vaccine as an immunising agent and that it may possess other serious disadvantages. In the circumstances it has been decided to discontinue the preparation of No. 2 vaccine at the Ministry's Veterinary Laboratory until further notice.

Obituary

RICHARD HUDSON, F.R.C.V.S., RETFORD

RICHARD HUDSON passed peacefully away at his home in Retford on Monday, November 20. He was 68 years of age. He had been in failing health for about two years, and, although he had undergone a major surgical operation, this failed to stay the progress of his illness. His courage at the time of operation and the stoical calmness with which he met his gradual decline in health was typical of the man. He continued to go about his work as usual until an increasing weakness and fatigue compelled him to take to his bed about seven weeks before the end. To his friends it was a great distress to see him gradually getting more and more frail. He still went to his patients, even when he had great difficulty in getting from his car. I saw him on several occasions during this period, but never heard a grumble; instead, he would discuss the progress of his trouble in the typical Hudson way of investigating a case. At the same time he never lost his great interest in his profession, and only as recently as September he had become interested in the Chromosomes of the Dog Daisy, for he thought that if he could add to his knowledge in these matters it might help him in his work connected with genetical problems in animals. Such

an outlook was typical of a man who throughout his life had neglected no avenue which might add to his store of knowledge.

He was born in Retford in June, 1876. His father and grandfather were veterinary surgeons in the town and, like Richard, they were graduates of the London College. It is interesting to note that his grandfather went to London by pack-horse, and on some occasions he would proceed to Hull by pack-horse and there board a coasting vessel bound for London. Richard's father died in 1887 at the age of 35. He left a widow and six children, of whom Richard was the eldest and aged eleven years.

His mother, who still survives, carried on the family practice until such times as Dick could qualify. She was a courageous woman and, fortunately, she had the good fortune to be able to employ good and competent veterinary surgeons as managers. They were T. Healy, J. G. Clayton and David Imrie. Each in turn took a great interest in Dick, and together they coached him for his future career. Hudson has told me how grateful he was to them and attributed his subsequent success in no small degree to their guidance. David Imrie probably moulded him more than the others, for many of us could recognise the Imrie touch in Hudson's life and work. He was educated at King Edward VI Grammar School at Retford and later proceeded to St. Augustine Grammar School at Dewsbury. From the latter he took the London Matriculation, no mean feat in those days, and then entered the Royal Veterinary College. His career there was good and he qualified at the age of twenty. One year later he was able to take over the practice which his mother had so nobly preserved for him. It was soon evident that the practice had come into good hands, for from the start he gave signs of that merit which was later to be recognised as genius. He became a F.R.C.V.S. in 1905. In 1902 he married Miss M. L. Rothwell, a native of his home town, and so began a happy home life which was to last for 42 years.

All through his life Hudson was a student and an original thinker. He became a great clinician and a most competent surgeon. Any task looked an easy one in his hands. He had great gifts and made full use of them. In many ways he reminded one of David Imrie, who had been one of his mentors. He always strived to know the truth about his cases, and to do this he read widely and took every opportunity to make P.M. examinations. In his endeavour to become an expert surgeon he spent many hours at the slaughter house, operating upon the freshly-killed animal until his hands and technique were perfect. He was always willing to give his services and knowledge to the aid of his fellow practitioners, so that he became the refuge, when they were in trouble, of all his neighbours. From time to time he contributed to veterinary literature, but he was probably at his best when giving a demonstration at some congress or society meeting. He loved to talk of his work, and those who were privileged to chat with him in his home would lose all count of time; such was the interest which in his quiet way he could enthuse. He was a born teacher and would have been an ornament on the staff of a veterinary college.

As to the man himself, he was a slightly built, modest and cultured man who read widely and was well informed in a great variety of subjects apart from those particularly appertaining to his profession. He loved all the simple

joys and things of life, the country, the villages, churches, and especially children and animals. He was a good mechanic and any spare time would be spent in his workshop, where he would on occasion fashion his own tools. This attribute to be able to fashion one's own tools is said to denote " a king amongst craftsmen." He had an orderly mind and lived up to the old motto which used to be displayed at the R.V.C., i.e., " Order is gain. A place for everything and everything in its place." He lavished the greatest care on his instruments, and they were consequently always in perfect order.

To go on his rounds with him was a joy; people were pleased to see him and his clients almost treated him with reverence. He had a kindly smile or nod to everyone. In his latter years he was troubled with deafness, but this did not upset the even tenor of his way.

He attained great honour in his own country and amongst his fellow practitioners.

The funeral took place at Retford on November 23 and the large number of people who attended to pay their last respects was in itself a great token of the esteem in which he was held. The profession was represented by Major T. Hicks (N.V.M.A. and Lincolnshire Veterinary Medical Association), B. Sampson (North Midland Veterinary Medical Association), and Messrs. T. Bowett, F. Talbot and T. Shanks. He leaves a widow and three children. The latter are Mr. J. R. Hudson, B.Sc., M.R.C.V.S., of Kenya, Mrs. J. Baxter Lechlade and Mrs. E. Hurst.

J.McC.

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